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PROCEEDINGS



FIRST WORKSHOP ON FIRE BLIGHT RESEARCH

COLUMBIA, MISSOURI
FEBRUARY 13-14, 1969

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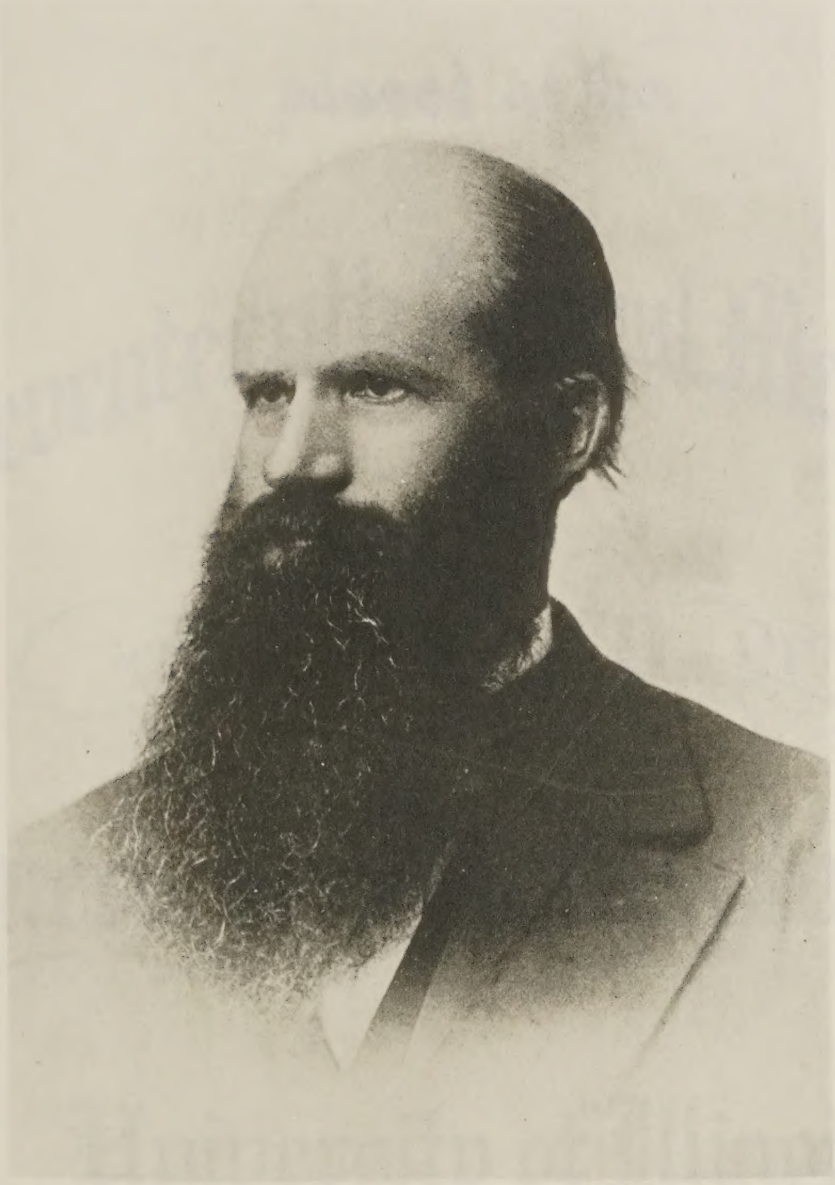
PROCEEDINGS

FIRST WORKSHOP
on
FIRE BLIGHT RESEARCH

held at Columbia, Missouri
February 13-14, 1969

UNITED STATES DEPARTMENT OF AGRICULTURE
Crops Research Division
Fruit and Nut Crops Research Branch

in cooperation with
University of Missouri
Department of Plant Pathology



Thomas J. Burrill

(1839-1916)

RESOLUTIONS

passed at the

Seventeenth Annual Meeting

OF

THE Society OF American Bacteriologists

University of Illinois

URBANA, ILLINOIS

December 28-30, 1915

WHEREAS

It is rarely possible for a scientist to make a discovery of such fundamental importance that it serves to develop an entirely new branch of science, and

Whereas

One of our hosts at this time,

Dr. Thomas J. Burrill

made such a discovery when he worked out the cause of pear blight and thus founded the science of bacterial plant pathology,

Therefore be it

RESOLVED

That the Society of American Bacteriologists regard it as a peculiar privilege to congratulate him for his pioneer and epoch-making work, and express its appreciation of his vigorous, enthusiastic and inspiring address of welcome,

Be it further resolved

That a copy of these resolutions be engrossed, signed by the officers of the Society, and presented to Dr. Burrill.

David Hendricks Berger.

PRESIDENT

John Weinzirl

VICE PRESIDENT

A. Parker Stitchens.

SECRETARY-TREASURER

Council

R. E. Buchanan.

H. J. Conn

W. A. Stocking Jr.

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FIRE BLIGHT RESEARCH WORKSHOP

A G E N D A

February 13

8:30 A.M. Introductory remarks

9:00 - 9:45 A.M. THE FIRE BLIGHT PATHOGEN

E. A. Grula	Nutrition and cell division in a species of *Erwinia*
B. C. Smale	Biochemical study of intervarietal resistance of *Pyrus communis* to fire blight
R. N. Goodman	The ultra-structure of *E. amylovora*; factors influencing the virulence of *E. amylovora*

9:45 - 10:30 A.M. Discussion

COFFEE BREAK

10:45 - 11:30 A.M. EPIDEMIOLOGY OF FIRE BLIGHT

D. Powell	Factors controlling the forecast of blossom blight infection
N. S. Luepschen	Observations on fire blight epidemiology in Colorado
J. E. Crosse	Ecological factors influencing the growth of *E. amylovora* and associated bacterial species

11:30 - 12:15 P.M. Discussion

LUNCH

1:30 - 2:15 P.M. TREE NUTRITION AND PHYSIOLOGY

K. G. Parker	Effect of tree nutrition and soil moisture on fire blight development
A. L. Kenworthy	Nutrition and physiology of the pear tree in relation to fire blight susceptibility
H. L. Keil	Effect of soil amendments on fire blight development; recovery of E. amylovora from apparently healthy pear tissue

2:15 - 3:00 P.M. Discussion

COFFEE BREAK

3:15 - 5:00 P.M. Resume Discussion Session

February 14

8:30 - 9:15 A.M. CONTROL OF FIRE BLIGHT

T. van der Zwet	Role of injury and longevity of E. amylovora in fire blight development
D. L. Coyier	Inoculation of apple and pear roots with E. amylovora; control of fire blight with Bacticin
E. J. Klos	Various aspects of fire blight control

9:15 - 10:00 A.M. Discussion

COFFEE BREAK

10:15 - 12:00 A.M. Resume Discussion Session

****

Nutrition and Cell Division in a Species of *Erwinia*

E. A. and Mary M. Grula
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A. General Information: This organism was isolated at least 20 years ago at the University of Arkansas and identified as *Erwinia carotovora*. Periodically, we have run biochemical tests to identify the organism; it keys at different times either as *Er. carotovora* or *Er. aroideae* but one or two tests (H_2S production or optimum temperature for growth) are usually at variance with descriptions given in *Bergey's Manual*. Because the characteristics of the organism do not fit precisely with any species description, we have designated it as *Erwinia* species.

From 1950 to 1959, the organism was routinely cultured on Yeast Extract Veal Infusion Agar. Since 1959, it has been carried on Nutrient Agar with or without dextrose (1%). When initially tested in 1959, the organism would not grow in a dextrose-ammonium chloride - salts medium (dextrose at 0.017M; ammonium chloride at 0.0187M concentration as used for *E. coli*) even after extended incubation. In 1966, it was observed that the organism would grow slowly and to a limited extent when the ammonium chloride level was raised to at least 0.05M. Growth is both rapid and extensive when four-carbon compounds, particularly malic or aspartic acid, are employed as the carbon source.

Unlike *E. coli*, this organism contains low levels of glutamic dehydrogenase, but high levels of aspartase activity. Therefore, the high ammonia requirement probably reflects a dependence for utilization of nitrogen via primary synthesis of aspartic acid (aspartase) rather

than synthesis of glutamic acid (glutamic dehydrogenase). Strong repression of aspartase synthesis (about 95% decrease) occurs when dextrose is present in the growth medium.

Stimulation of growth by four-carbon compounds such as aspartic or malic acid probably occurs because the organism does not fix carbon dioxide to any large extent into either malic or oxaloacetic acid; rather greatest fixation of carbon dioxide occurs via synthesis of carbamyl phosphate. Because of the poor fixation of carbon dioxide into needed malic or oxaloacetic acid, synthesis of beta-alanine (via alpha decarboxylation of aspartic acid) limits the rate and extent of growth in many media. Feeding of either aspartic or malic acid negates the requirement for beta-alanine or pantothenate.

Citric acid can be used as sole source of carbon, but an extensive lag in growth occurs. Although none of the five-carbon intermediates of the Krebs' cycle can be utilized as sole source of carbon, all stimulate growth when dextrose is present; particularly alpha-keto-glutaric acid. The need for compounds such as dextrose to generate energy to move the five-carbon compounds into the cell is suspected.

Other characteristics of Erwinia sp. are as follows:

a) Fructo-aldolase activity can not be demonstrated therefore, existence of the Embden-Meyerhoff pathway is in doubt.

b) The organism possesses a potent glucose-6-phosphate dehydrogenase activity which strongly implicates operation of the oxidative pathway for glucose catabolism.

c) Isocitrate lyase activity cannot be demonstrated. Further, lack of growth on two-carbon compounds effectively rules out presence of the glyoxylate by-pass.

d) Oxygen utilization on Krebs' cycle intermediates (Warburg apparatus) indicates operation of the cycle. Also, all enzymes of the cycle have been demonstrated.

e) Phospholipids of the cell envelope are phosphatidyl ethanolamine (90%) and phosphatidyl glycerol (10%).

f) Ethanolamine may also be present in the lipopolysaccharide portion of the outer cell wall.

g) A thin mucopeptide layer is present in the cell wall. Growth in a dextrose-aspartic acid-salts medium yields a mucopeptide having the following components: Muramic acid-glucosamine-alanine-glutamic acid-diaminopimelic acid-aspartic acid-glycine. Molar ratios are: 1-1-2-1-1-0.2-0.05. Isolated mucopeptide is degraded by lysozyme; however, the mucopeptide is not attacked by lysozyme in the intact cell or cell wall.

h) Mesosomes are not present.

i) The organism divides by constriction (concomitant pinching of the cell membrane and wall). Formation of a membranous septum does not precede wall ingrowth or invagination. The division process requires energy.

B. Characteristics of Non-dividing Cells: Cell division is inhibited in a dextrose-aspartic-salts or a aspartic-salts medium by any of 6 D-amino acids (serine, phenylalanine, methionine, threonine, tryptophan or histidine), vancomycin, D-cycloserine, 5-fluorouracil, aminopterin, mitomycin c UV light, S-dichlorovinyl-L-cysteine or penicillin with the side chain modified in eight different ways.

Generally, prevention or reversal of division inhibition can be effected using pantoyl lactone, omega-pantoyl lactone, spermine or hypertonic conditions (organic or inorganic). These compounds must

be present continuously to cause division of filamentous cells. Thus, their effects appear to be more physical than metabolic. One notable exception has been observed, i.e., hypertonic levels (0.27M) of sodium or potassium chloride, further enhance division inhibition caused by penicillin. D- or L-Alanine L-glutamic acid, alpha-keto-glutarate, malic acid, certain divalent metal ions, or ammonium chloride can prevent inhibition of cell division when D-serine is the inhibiting agent; however, the effectiveness of any of these compounds depends on composition of the growth medium.

Normal size of these cells is 2 to 6 microns; however, depending on composition of the growth medium, filamentous cells can be routinely produced that are greater than 300 microns in length. Filamentous cells do not contain internal membranous septa; they can readily be induced to divide by compounds such as pantoyl lactone. We question if a critical cell length exists wherein division activity cannot be induced. The "short" critical length of these cells is about 0.85 microns (aspartic acid medium with high levels of pantoyl lactone). Size of cells must always be defined in terms of the composition of the growth medium.

Filamentous cells are leaky, i.e. they lose proteins, nucleotides and sodium. They do not lose RNA or DNA. On a dry weight basis there is no inhibition in synthesis of RNA, DNA or protein.

Filamentous and normal cells grow from both ends; however, the extension of growth from both ends is not always uniform.

When filamentous cells are stimulated to divide by first removing the inhibitor followed by inoculation of these cells into a complex growth medium, division is primarily restricted to the ends of the cells where new growth has occurred. When division of filamentous

cells is triggered by pantoyl lactone, a cell may divide anywhere along the length of the filament as well as at the ends.

D-Serine can inhibit cell division under either aerobic or anaerobic growth conditions.

When too high a concentration of a division inhibitor is used, viable growing cells will spheroplast and lyse rather than continue growth in a filamentous form. Spermine, but not pantoyl lactone, will protect the spheroplast form.

D-Serine behaves as an analog of L- or D-aspartic acid in that it inhibits both the uptake and endogenous alpha-decarboxylation of aspartate. Washed filamentous cells also exhibit impaired uptake of aspartate but not of dextrose. Uptake of aspartic acid is not aided in cells growing in the presence of D-serine when pantoyl lactone, spermine or hypertonic conditions are also present to allow cell division. Observations of this type help explain progressive inhibition of growth by increasing concentrations of D-serine and also help explain general lack of growth stimulation when pantoyl lactone, spermine or Carbowax 400 are used to prevent cell division inhibition by D-serine.

D-Serine enters into the mucopeptide (about 1.0 molar amount compared to alanine). This aberrant incorporation does not alone account for inhibition of division because the amount of serine incorporated into mucopeptide is not decreased when division inhibition by D-serine is prevented by pantoyl lactone or other agents. Several types of filamentous cells (D-serine, vancomycin, penicillin, UV, mitomycin c, cycloserine grown) contain a reduced amount of mucopeptide (30 to 40% reduced). This reduction in mucopeptide deposition also is not corrected by most compounds or conditions which induce division activity.

Secondary membrane damage (leakiness and impaired uptake of aspartate) occurs; however, ratios of cell phospholipids are not altered and qualitative changes in phospholipid composition have not been detected.

Conventional staining procedures coupled with optical microscopic observation reveals that filamentous cells possess discrete separated nuclear bodies running the entire length of the cell. Electron microscopic observation of thin slices (serial sections), however, reveals that although the nuclear body continues to grow and contains normal appearing amounts of DNA, the nuclear body is not dividing (mitomycin c, penicillin and D-serine grown filaments). We have therefore concluded that both cell and nuclear body division are inhibited during growth into the filamentous form. We have also observed that when filamentous cells are triggered to divide by pantoyl lactone, division of the nuclear body quite often does not precede obvious initiation of cell division activity (observable invagination of the cell membrane and wall). Such observations have also been made using normally growing and dividing cells.

Quantitative changes in various enzyme activities have been consistently observed in D-serine grown filaments. Greatest change observed thus far has been in activity of malic dehydrogenase. Depending on composition of the growth medium and age of cells, increases of 2 to 9 fold have been routinely observed.

Although filaments are not usually susceptible to plasmoptysis in distilled water, the ability of cells to take up oxygen is completely annulled on substrates such as glucose or pyruvate after just one freezing and thawing. Normally growing and dividing cells are little affected regarding oxygen uptake after similar treatment.

C. Current Activity: Four recent observations will be discussed:

1. In a dextrose-salts medium, poor growth results when the level of ammonium chloride is 0.015M; better growth occurs when the ammonium level is raised to 0.09M. Cell size is 2 to 4 microns under both conditions. If D-serine is added to these media at the time of inoculation, cells grow into the filamentous form at the low concentration of ammonium; however, at the high concentration they divide normally even though D-serine limits the rate and extent of growth. Thus, high levels of ammonium ion prevent inhibition of division by D-serine in dextrose-salts medium.

2. Addition of pantothenic acid or beta-alanine ($2X10^{-5}$M) in the presence of D-serine in the dextrose-salts-high ammonia medium overcomes greatly growth inhibition; however, instead of normal sized cells, very large filaments are now formed. Under these growth conditions, pantothenic acid behaves as a potent inhibitor of cell division.

3. D-Serine inhibits uptake of aspartic acid; it also decreases synthesis of beta-alanine by inhibiting the alpha-decarboxylation of aspartic acid. Action at these two levels (exogenous as well as endogenous and involving the aspartic acid molecule directly) indicates that D-serine behaves as an analog toward aspartic acid. Further testing of this hypothesis by nutritional experiments has shown that the toxicity of D-serine in a aspartic acid-salts medium can be significantly reduced by addition of inosine (for purine synthesis), dihydroorotic acid (for pyrimidine synthesis), diaminopimelic acid (for mucopeptide synthesis) and beta-alanine (pantothenate and Coenzyme A synthesis). Effects of these compounds on size is also significant; however cell size is never completely normal. Addition of glycine and ammonium chloride with these compounds allows for nearly complete reversal of D-serine toxicity on both

growth and cell division. Therefore, it appears that D-serine is an analog of aspartic acid and L-serine (inhibits removal of hydroxymethyl group from L-serine to allow formation of glycine). A precise role for ammonia is not yet apparent.

4. If D-serine is present from time of inoculation in a dextrose-aspartic-ammonia-salts medium growth is decreased and relatively long cells form. If, however, D-serine is not added until about 12 hours after inoculation spheroplasting is initiated and complete in 99.9% of all cells within a 24-hour period. If our initial experimental protocols had been set up in this manner (addition of D-serine to exponentially growing cells), inhibition of division would never have been observed; instead D-serine would have been classified as a spheroplasting agent. These spheroplasts are unique; they are stable in the growth medium in the absence of osmotic protection yet they are completely lysed in distilled water.

Ability of the cells to undergo this spheroplast transformation is nutritionally controlled. If either ammonium chloride or dextrose is left out of the medium, spheroplastswill not develop; instead, completely lysed rod ghost forms develop.

Publications relating to Erwinia species

Journal of Bacteriology:80:369(1960); 80:375 (1960); 83:931 (1962); 83:492 (1962); 84:599 (1962); 90:1054 (1965).

Canadian Journal of Microbiology:11:605 (1965); 11:453 (1965); 14:295 (1968); 14:1217 (1968); 14:1225 (1968).

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Journal of General Microbiology:41:155 (1965).

Biochemica Biophysica Acta:74:776 (1963).

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Our research has been supported by The National Science Foundation (Grants GB-9848, 259, 1532, 4357 and 5641) and The Research Foundation, Oklahoma State University (State Project #91).

A Biochemical Study of the Intervarietal Resistance of Pyrus Communis to Fire Blight

B. C. Smale[1/]

The mechanism responsible for fire blight resistance in leaves of 6 commercial varieties of Pyrus communis appears to be wholly a host reaction, activated by mechanical damage to host tissues. The following factors were associated with leaves of highly resistant varieties: (1) presence of large amounts of arbutin and free hydroquinone in unaltered leaf homogenates; (2) accumulation and persistence of antibacterial concentrations of hydroquinone enzymatically released from arbutin during oxidation of leaf homogenates; and (3) disappearance, following cell disruption, of an unidentified antagonist of hydroquinone. The balance between the rapidity of the appearance and disappearance of antibacterial hydroquinone in mechanically damaged leaf tissue is a major determinant of relative blight resistance.

The extension of this study to other Pyrus species and P. communis varieties did not give such a clear cut relationship. The mechanism responsible for blight resistance in Pyrus is apparently the result of a balance between host and pathogen physiology mediated by environmental conditions.

Reference:

Smale, B. C. and H. L. Keil. 1966. A biochemical study of the intervarietal resistance of Pyrus communis to fire blight. Phytochemistry 5: 1113-1120.

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The Hypersensitive Reaction and Its Possible Relationship to Pathogenesis in Plants Infected by Bacteria, with Special Reference to the Fireblight Bacterium.

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The hypersensitive reaction (HR) is a defense reaction of plants against species of pathogens which normally do not parasitize them (incompatible host-parasite relationship). When pathogens attack such "uncongenial" hosts (normally non-susceptible species) or resistant plants (normally susceptible species) the plants react with a rapid cellular collapse and the development of tissue necrosis, the symptoms of HR.

Hypersensitivity of plants to virus and fungus pathogens has been known for some time (11). Klement and Lovrekovich (9) clearly demonstrated the existence of HR in plants to bacterial plant pathogens. Using the syringe-tobacco leaf infiltration technique devised by Klement (6), it is possible to evoke cellular collapse as rapidly as 6 hours with complete necrosis and desiccation of the infiltrated tissue in 18 hours (2).

Experimentally, the reaction is induced in either the uncongenial host or resistant plant by infiltrating leaf tissue with 5×10^6 or more bacterial cells/ml. Infiltration with fewer than 10^6 cells/ml generally results in small isolated lesions which are visible only with the aid of a hand lens (8).

The most revealing characteristic of HR as it develops in plants inoculated with pathogenic bacteria is the rapidity with which host tissues lose their turgor (6 hr in the uncongenial host-parasite combination of tobacco and Erwinia amylovora). Since loss of turgor is quickly followed

(within 24 hr) by complete desiccation of the affected leaf tissues it would appear that a profound change in permeability of host cell membranes (plasmalemma and/or tonoplast) occurs.

The sensitive site of the membrane, which may be altered, was assumed to be either the protein component, the phospholipid component or both (8).

Recently, conclusive experiments in our laboratory indicated that phosphatidase activity, which increases during the course of HR development induced by E. amylovora, is not causally related to HR (5). For example, infiltration of tobacco leaves with 10^6 cells/ml of E. amylovora which does not cause visible HR, significantly increases phosphatidase activity. In fact, the level of increased enzyme activity is equal to that detected in tobacco leaf tissue infiltrated with either 10^7 or 10^8 cells/ml of E. amylovora. The two higher concentrations of bacterial cells cause HR symptoms to develop in 6-8 hr. This and other evidence (4), strongly suggests that HR does not occur as a result of any perturbation of the phospholipid component of vital cell membranes.

That the membrane component which is altered is the protein component as suggested by experiments wherein small sulfhydryl-containing (SH) compounds are infiltrated into tobacco leaves (2). The data reveal that both SH-containing compounds, mercaptoacetic acid and mercaptoethanol induce electrolyte leakage from infiltrated leaf tissue. It is apparent that their ether analogs (-S-) thiodiacetic and thiodiethanol, at 4 times the concentration of the mercaptans cause no leakage at all. This leakage provides direct evidence that the selective permeability of the vital membranes of the cells has been lost. It should be mentioned that the time at which electrolyte leakage is detected is precisely the time when the early visual symptoms (tissue collapse) of HR became apparent. That membranes are indeed

being affected at the time electrolyte leakage occurs has been established electronmicroscopically. Our electron micrographs indicate significant damage to chloroplasts, primarily to the bounding membrane (envelope) of that organelle. The distortion of the grana and inter grana lemellae seems similar whether induced by bacteria or SH-containing compounds. We have proposed therefore that the membrane component being altered is the protein and that this is being accomplished by reduction of S-S bonds to -SH (2, 8).

That HR may be a partial manifestation of pathogenesis *per se* was first considered as a result of data obtained when apple leaves were infiltrated under vacuum with either an avirulent and virulent strain of *E. amylovora* (1). It is apparent that both strains grew equally well in leaf tissue and both caused electrolyte leakage and leaf necrosis. It may also be unequivocally stated that the avirulent strain was unable to induce *any* of the symptoms associated with the fireblight disease when inoculated into stem (7) or pear fruit tissue (3).

Since both the virulent and avirulent strains induced symptoms indicative of HR their effect upon cellular ultrastructure was examined in the electron microscope (4). It is apparent that both strains cause membrane damage. Clearly, the bounding membrane of the chloroplast is disrupted, mitochondria are absent at 24 hr after inoculation, cytoplasm becomes aggregated and the plasma membrane separates from the cell wall. These intra-cellular alterations are similar to the ones which occur in tobacco leaves infiltrated with *E. amylovora*, (an obvious incompatible combination).

Two explanations may be offered for the similarity of symptoms in apple leaves induced by the virulent and avirulent strains of *E. amylovora* (1).

It is possible that apple leaves are not particularly "susceptible" host tissue, for example, not as susceptible as either apple stem apices or young (green) pear fruit (3, 7). Hence, the symptoms induced by either strain is strictly HR and not normal disease development.

More plausible at this time is the possibility that membrane and organelle disruption, leakage of electrolyte, tissue collapse are general responses of leaves to plant pathogenic bacteria (4). Further, the intensity of the response is governed by the size of the inoculum (1). We have recently observed that if apple leaf tissue is infiltrated with either virulent (compatible) or avirulent (incompatible) strains of E. amylovora and the infiltrated leaves remain exposed to the atmosphere, typical disease symptoms will develop in the case of the former and HR in the case of the latter. However, if the infiltrated tissue is maintained under conditions which do not permit the water leaking into intercellular spaces to be lost to the atmosphere (e.g., in humid chambers) (1) then the typical HR (desiccation) will not occur and the water soaking of leaves (a symptom of pathogenesis) develops in leaves that have been inoculated with either the virulent or avirulent strain of the pathogen.

The apparent pathogenic activity of incompatible bacteria in water congested plant tissues (e.g., potato tuber) long been recognized however, the precise nature of this phenomenon remains unknown (10). What is that the bacterium does per se to cause the cell membrane to deteriorate is currently the object of intense study in our laboratory.

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Annual distribution of 1200 publications on fire blight during the period 1870 - 1970.

PREDICTING THE BLOSSOM BLIGHT PHASE OF THE FIRE BLIGHT DISEASE ON APPLE IN ILLINOIS

Dwight Powell [1/]

The twig blight phase of the fire blight disease caused by Erwinia amylovora (Burrill) Winslow et al, normally conforms to a pattern by responding to adequate rainfall and optimum temperatures. Blossom blight, however, is much more erratic, especially on apples, and has not always developed even though in-bloom temperatures and moisture conditions were optimum.

Thus, the development of blossom infection was studied closely for approximately 10 years, from 1952-1961. Fifteen different orchards were selected which had a history of fire blight. These were located within an area from 38 to 42° latitude; within a range of 3 degrees longitude; and represented four isothermal zones. Each orchard was supplied with a recording thermometer and rain gauge. The grower recorded daily rainfall on a prepared chart.

Observations were made weekly except an extra visit during blossoming. Fire blight blossom infections were made during such visits on the Jonathan cultivar. A 10% blossom infection was considered severe.

A correlation appeared to exist between blossom infection and prebloom freezing temperatures (4).

In discussing the results, "early bloom" means the opening of the first (center or king) blossom of a cluster. Minimum temperature for the growth of the pathogen is 65° F (2,3). "Degree days" are the degrees which accumulate from day to day above 65° F between the latest prebloom freeze and the "early bloom" period.

1/ Plant Pathologist, University of Illinois, Urbana, Illinois

When blossom blight was severe the maximum in-bloom temperatures were in a range of 76° F, the optimum for growth of the fire blight pathogen (2,3). The degree days which accumulated prior to early bloom totaled more than 30 in the different orchards. Rainfall was adequate, but not excessive during the bloom period.

In some orchards in which blossom blight did not occur in-bloom temperatures and moisture appeared to approximate the optimum. In these orchards, however, prebloom freezing temperatures occurred immediately preceding early bloom. The degree days had not accumulated to 30 in these orchards.

Other orchards did not develop blossom blight. These did not have prebloom freezes and degree days had accumulated to above 30. Weather factors thought to have inhibited infection in the various orchards were daily low in-bloom temperatures, drought, excessive in-bloom rainfall, and daily maximum in-bloom temperatures above 86° F(1).

Thus, in predicting blossom infection, the only additional factor to those already known is the prebloom freeze.

To summarize, conditions of weather leading to blossom infection are:

1. Temperature
    a. At least 30 degree days (above 65° F) between the latest prebloom freeze and early bloom.
    b. Maximum temperatures between 70-80° F during early bloom.
2. Moisture
    a. Adequate rainfall preceding bloom so that normal growing conditions prevail.
    b. Light, occasional rains with high humidity (70% +) during early bloom.

Conditions interfering with blossom infection are:

1. Temperature
   a. Freezing temperatures close to early bloom. Less than 30 degree days (above 65° F) between latest prebloom freeze and early bloom.
   b. Maximum temperature either lower than 65° F or higher than 86° F during early bloom.
2. Moisture
   a. Drouth preceding and during early bloom(1).
   b. Excessive rains during early bloom.

Using these criteria, blossom infection predictions have been made for 6 years through a weekly spray service report. Information for the report is obtained by weekly calls to key fruit growers in the different thermoclinal areas. Thus far, the information has been correct. The use of streptomycin in bloom is determined by these predictions.

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Observations on Fire Blight Epidemiology in Colorado

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Fire blight of Bartlett pear trees is an economic problem for Colorado growers despite the fact that ours is a semi-arid climate with only 7 - 12 inches annual precipitation. A trace of fire blight is present nearly every year, enough to supply well distributed inoculum potentially able to cause an epiphytotic. The latest severe outbreak in Mesa county, where 70% of our pears are grown, was in 1966, when 5% of our trees were lost and much scaffold wood destroyed. Blight in 1967 was about half as severe. In 1968 there were few new infections. In Delta county, at altitudes of 5,000 - 7,000 ft., blight infections can be found in all areas nearly every year, due to later bloom periods and more shower activity.

Blossom blight during main bloom usually accounts for a small percent of our blight problem, often just enough to provide secondary inoculum. Cool weather and frosts during bloom often result in maximum temperatures well below 65°F. In Mesa county rain showers are infrequent, morning dew is very light, hail storms at this time are occasional and very local. Summer conditions are very warm and dry. Under such conditions I was very surprised to find the incidence of blight that occurred. Also, shoot and limb blight of severe proportions is seldom found once Bartletts are over 15 years old.

The most common entry points for blight infection are (1) terminal or "rat tail" bloom, (2) succulent terminals, and (3) green fruit. Terminal

bloom develops throughout the growing season. Heavy nitrogen fertilization and/or frequent irrigations may stimulate two or more flushes of succulent growth. Frequently, active blight development with ooze can be found on Bartlett shoots and limbs as late as October 1.

Green apple aphids appear to be the main summer vectors, while Lygus bug and rosy aphids also are suspect at various times. Leafhoppers and psylla are not a problem on pear in Colorado. Occasionally we find a spread pattern suggesting splashing rain dispersal within a tree during main bloom, and there are isolated cases where blight follows hail damage. Fire blight development in Colorado is dependent on favorable temperatures, but correlations with precipitation and relative humidity cannot be determined very readily. This suggests that sucking insects are successfully transferring inoculum into pear tissue completely independent of atmospheric moisture conditions.

Fire blight on Jonathan and Rome apples is generally of less economic importance, except in very vigorous young plantings. In mature apple trees blossom and twig blight reduces the yield somewhat, but seldom progresses into the scaffold. Frequently infected oozing fruits can be found on the trees, with the blight spreading down through the pedicle into the supporting spur.

Streptomycin sprays during bloom are now being used with some success by many Colorado growers, whereas in the past zineb and copper materials were utilized, with very limited control. Fixed copper is recommended in cover sprays but its effectiveness during the summer has not been determined under our conditions. Some pear growers have been checking blight

by using 1 or 2 streptomycin applications at 200 ppm late in bloom or early summer. However there is still much room for improvement in the use of streptomycin: e.g. spray timing for optimum protection, and improved absorption. The use of zinc and cadmium canker paints has been encouraged the last few years, with some cases of great success. Summer cutting has been disappointing and uneconomical. Withholding of midseason and late irrigations can check blight severity in some cases.

Since streptomycin is the most effective material against fire blight (and hoping that label restrictions would be changed) we tried in 1967 and 1968 in a limited effort, to improve cuticular penetration under our low R.H. conditions. Bartlett pear trees were sprayed with streptomycin - surfactant combinations in the orchard during daytime conditions. Shoots were harvested, brought into the lab, placed in jars of water, and the terminals inoculated with Erwinia amylovora by hypodermic. Results of the 1967 study are shown in the following table. In 1968 various factors interferred with our test, but the strep-Tween 20 combination appeared to be the best.

Percent Fire Blight Control - Detached Pear Shoot Study, 1967*

Treatment	Experiment No. I (July 25)	II (July 31)	III (Aug. 10)
100 ppm strep. (Agri-Strep)	52%	21%	0%
200 ppm "	73	54	0
200 ppm " + ½% Tween 20	84	71	75
" " " + ½% Glycerol	84	43	21
" " " + ½% Atlox 209 (50%)	65	56	13
" " " + ½% Buffer-X	52	60	0
" " " + ½% Biofilm	51	59	54
" " " + ½% Atlox 1087	57	53	33
" " " + ½% ACL 574 (50%)	—	40	17
" " " + ½% ACL 577 (25%)	—	53	50

* Control data - mean of 10 shoots, compared with 10 controls/expt.

AN ENRICHMENT TECHNIQUE FOR ERWINIA AMYLOVORA

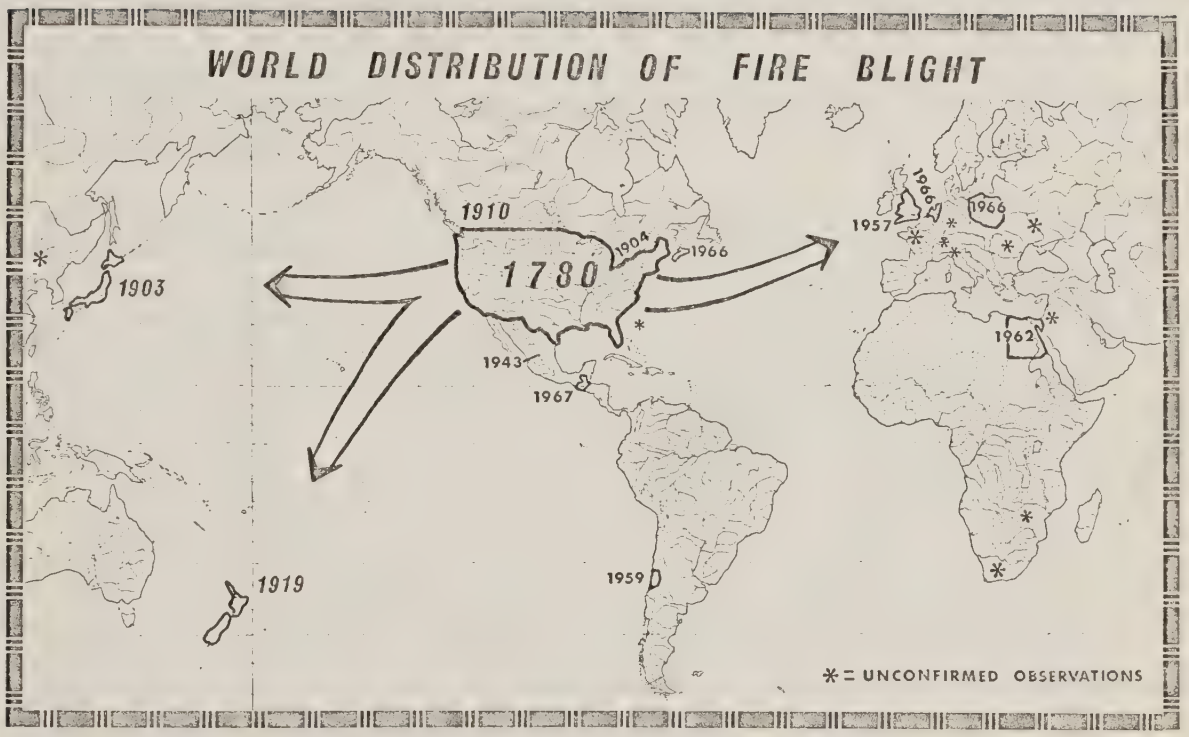
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The isolation of specific organisms by means of differential and selective media has been a routine procedure in medical bacteriology for many years, but these techniques have so far found little application in plant bacteriology. We are currently experimenting with a selective medium for the enrichment culture of Erwinia amylovora. It is hoped that this will facilitate studies on the epidemiology of fireblight, in particular the detection of small populations of E. amylovora on the host surfaces and in other habitats.

The basis of the medium is 40% (w/v) sucrose in nutrient broth. E. amylovora grows well at this sugar concentration but the majority of saprophytic bacteria are inhibited. The selectivity of the medium has been further enhanced by the inclusion of (1) crystal violet to suppress the Gram-positive bacteria and of (2) potassium dichromate to inhibit E. herbicola and a few other Gram-negative forms capable of growing in 40% sucrose.

In tests to measure the selectivity of the medium, leaves and soil with their attendant microfloras, were incubated in the broth in the presence of small inocula of E. amylovora (less than 10 cell/ml). In most cases E. amylovora was enriched to the extent that it became the dominant organism in 3-4 days. Not infrequently the pathogen was recovered from the broth in pure culture.



Dates of earliest known observations of fire blight in 11 countries; and locations of unconfirmed recordings of blossom blight in 12 other countries.

Arrows do not necessarily indicate routes of dissemination of the fire blight pathogen.

(from: Plant Dis. Reptr. 52: 698-702, 1968)

FIRE BLIGHT: SOIL MOISTURE AND TREE NUTRITION

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On the basis of many years observation in the orchard, Blake (1) recommended that susceptible varieties of apple and pear be planted on deep, fertile, and well-drained but not droughty soils. He suggested that anything which will restrict the root system or its function will result in greater susceptibility and that such a condition may result where growth is slow and weak as well as excessively rapid because of low carbohydrate content of the tissue in either case. Also, Blake (2) reported severe outbreaks in seasons following unusually dry conditions the previous summer.

In a survey of pear orchards Fisher et al. (3) found less severe fire blight infection in orchards on well-drained soils than on poorly-drained ones. On trees growing in soils intermediate in drainage the disease was intermediate in severity. In an experiment on relation of cultural treatment to disease development, inoculations (made by Dr. N. S. Luepschen) to Bartlett pear shoots detached from orchard trees and maintained in sand under mist gave more extensive invasion in shoots from trees in the poorly drained part of the orchard than in a better-drained part. Counts made at the end of June on terminal bud formation (5) showed growth stopped earlier in the well-drained part, despite the fact that growth and yield were better in that part of the orchard. These results have been supported over the years by numerous observations on disease development in commercial orchards, both pear and apple. Available nitrogen level in the soil in mid-summer was higher in the poorly-drained soil than where drainage was better. These data were not published because of the untimely death of Dr. Fisher.

Hildebrand and Heinicke (4) made measurements on fire blight development on apple trees that received different cultural treatments. Greater numbers of blossom and shoot infection occurred under cultivation and under alfalfa culture than under grass sod. Less extensive infection, however, as measured by invasion of larger branches, occurred in the trees under alfalfa culture than under either of the other two treatments. That is, despite larger numbers of infections under alfalfa than under grass sod total damage to the trees was less. No determinations of soil moisture were reported but it is expected that under alfalfa cover crop moisture levels in the root zone of the trees would be lower than under grass sod or cultivation.

Tree Nutrition

It is generally considered that rapid highly succulent growth favors blight development, at least in part explained on the basis of high vapor-tension in the tissues which Shaw (6) found favored invasion of the tissues by the pathogen. However, many orchards exist which are in moderate to good vigor and produce good crops without a severe blight problem. Blake's suggestion that naturally fertile soils are favorable indicates a well-balanced nutrition may be important. In the survey (3)

"some association was found of high levels of soil phosphorus and potassium with decreased fire blight severity, though the data were not significant statistically".

Two experiments were made in Bartlett pear orchards in a factorial design of 2-tree replications, applying N-P-K in one case and mulch-N-P-K in the other (3,5). The orchards were in sod. The +P treatment was 20 pounds of superphosphate applied in the fall, 1953; +K was 5 pounds of muriate of potash in the spring of 1954 and 7 pounds of sulfate of potash in the spring of 1957. The +N treatment was 2 pounds of ammonium nitrate applied each spring. In addition, the growers applied 1 1/2 to 2 pounds of ammonium nitrate to all trees each spring, except in the Horn Farm experiment which received urea foliar sprays instead the first 2 years, 1954 and 1955, the light ground application of ammonium nitrate in 1956 through 1958 plus supplemental urea sprays as needed, and no overall nitrogen application in 1959.

In one experiment (Horn Farm) considerable natural blossom infection occurred in 1959, and severity was rated on each tree by measuring the extent of each lesion and determining the average for all infections on the tree. Results (5) indicated extra nitrogen increased fire blight susceptibility. The addition of potassium with nitrogen caused no increase in susceptibility. The addition of phosphorus with extra nitrogen increased susceptibility, and the most severe disease was where both phosphorus and potassium were added with extra nitrogen.

In the second experiment (Sodus Fruit Farm) a similar series of treatments was made, with straw mulch added to the factorial. One bale of straw was spread around each +mulch tree in 1953, 1954, and 1957. During the period of the experiment few natural infections occurred, inadequate for any rating of the trees for susceptibility. Susceptibility was tested by inoculating detached shoots, already described in the section on Soil Moisture. Results are described by Fisher, et al. (3) and Parker et al. (5).

Mulch applications increased blight development as measured by length of invasion on inoculated detached shoots. Nitrogen content of leaves was increased on trees with added mulch. There was a tendency (not statistically significant) for blight infection to be decreased with potassium additions; in this orchard potassium in leaves on untreated trees was below the optimum. Phosphorus gave no apparent influence on disease development.

In a study on sand culture, made by Dr. Luepschen (5), the results generally support those in the field. The addition of potassium to nitrogen had little influence on blight development, but the addition of phosphorus or phosphorus and potassium to nitrogen increased disease development.

Discussion

At the Sodus Fruit Farm, where there was a response in yield and fruit size to potassium treatment there was an apparent reduction in disease on trees receiving potassium. At the Horn Farm, with potassium adequate for growth, potassium additions did not influence disease, except where phosphorus also was added. Phosphorus additions did not influence disease at the Sodus Fruit Farm, where the natural level of this element

was at least somewhat deficient. In the Horn Farm experiment, where the natural level of phosphorus was higher, this element increased disease when added along with extra nitrogen.

It is suggested that phosphorus should not be added unless a need for it is clearly established, but that potassium should be added unless it is clear a growth response cannot be expected. Probably potassium in excess might be dangerous because of antagonism to calcium and other elements. Mulch should ordinarily not be used except under droughty conditions, and where used must be taken into account in any potassium treatments.

Soil drainage appears to be a key to providing a basis for improved growth without increased susceptibility to fire blight. Pears are generally grown on poorly-drained soils in New York State, not because they grow better under such conditions (they do not) but because they will survive where other fruit trees die. It is recommended that our growers select well-drained sites for pears, and they are encouraged to provide drainage where this seems needed and the possibility exists that drainage can be improved economically. They are encouraged to maintain potassium at standard levels, but it is suggested they do not add phosphorus unless they are sure it is clearly needed. It is recommended that pH be maintained at 6.0 to 6.5 from the standpoint of maintaining a balanced nutrition and to ensure the availability of adequate calcium. Liming was not included in the experiments butthe pH was near the suggested level in each case, probably because of overall liming shortly before the experiments were initiated.

Because early cessation of shoot growth appears to be desirable, Dr. G. H. Oberly suggests that nitrogen be applied early and that a nitrate form be used so that available nitrogen will be depleted early in the summer. Also, it is recommended that such practices as annual pruning be followed so that only small cuts need be made to avoid the stimulation of excessive sucker growth. Where large cuts must be made or severe blight infection occurs reduction in nitrogen application is advised the following year.

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2/10/69

Nutrition and Physiology of the Pear Tree in Relation to Fire Blight Susceptibility

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Most of the reports regarding the susceptibility of pear (and apple) trees to fireblight as related to nutrition and soil management are based on secondary evidence rather than results of experiments planned with the specific objectives to study the relationships. From such observations has developed the concept of increased nitrogen levels (or vigor) decreasing resistance to fireblight.

Unfortunately (for the industry), this has been interpreted into the practice of withholding nitrogen as a means of fireblight control. In many instances, nitrogen has been withheld to the point of very low production of very poor quality fruit of small size. In Michigan, when we have convinced certain growers that nitrogen applications are needed to stay in production, we seem to have a discouraging year of heavy fireblight. At present, several of our growers are sufficiently discouraged to remove their pear trees.

I would like to refer to an experiment conducted in 1958-60, to make a specific study of the relationship of tree nutrition to fire-blight susceptibility. The graduate student (now Dr. Lowell Lewis, Riverside, California) started with a statement that he found in the literature to the effect that asparagine was required for growth of the

fireblight organism. He reasoned that if he could accomplish reasonable control of asparagine levels in the tree he should accomplish increased resistance to fireblight.

One-year-old Bartlett pear trees on a French seedling rootstock were grown in sand culture using 23 different solutions (see column 1, Tables 1 and 2). The trees were grown one year and leaf analysis made to determine the nutrient levels obtained. They were then over wintered and grown the second year and third year. On the third year, the trees were sampled for amino acid analysis of shoots and leaves and inoculated with fireblight organism to determine susceptibility.

Table 1 shows nutrient levels achieved as reflected by leaf analysis, total amino acids in shoots and the susceptibility index.

Leaf composition values show that deficiency levels were obtained for N, P, K, Ca, and Mg. The minor element treatments, however, did not show consistent symptoms. Only the level of Mn was equivalent to a deficiency. The high levels did not show any clearly defined toxicity symptoms. The high N trees had a reduced amount of growth and the leaf composition value for N was much higher than ever observed for field grown trees. The high K trees showed a symptom similar to Mg deficiency; the Mg level for these trees was 0.08%.

The data for total amino acids shows a distinct influence of nutritional environment. Low N, Ca and Mg resulted in a reduced level of amino acids. An increase in Amino acids resulted from high N, P and K. Other treatment effects could not be considered as significant.

Table 1. Bartlett pear trees. Nutrient status, total amino acids, and Index of susceptibility to fireblight.*

Nutrient Level	Leaf Composition			Total Amino Acids	Susceptibility Index
	Treatment	Control	Standard		
Control				100	58
0.2 N	1.34			44	93
3 N	3.28	2.06	2.33	188	169
- P	0.07			98	31
5 P	0.55	0.13	0.23	192	86
- K	0.49			102	86
5 K	4.68	1.65	1.53	224	175
- Ca	0.44			80	127
5 Ca	1.03	0.83	1.40	98	--
- Mg	0.04			46	106
5 Mg	0.37	0.24	0.41	102	156
- Fe	137			66	55
15 Fe	544	167	220	96	115
- Mn	14			94	135
25 Mn	811	29	98	72	93
- B	13			76	--
15 B	155	29	42	42	86
- Cu	7			90	154
25 Cu	11	7	23	102	127
- Zn	24			104	124
15 Zn	35	26	30	104	181

* Leaf composition as % for N, P, K, Ca, Mg and ppm for Fe, Mn, B, Cu, and Zn. Standard values used for diagnostic work. Total Amino acids in shoots as micrograms/100 mg fresh wt. Susceptibility value calculated as sum of % inoculation infections plus % of wood infected.

The susceptibility index showed a decreased susceptibility for low P. The inoculations were not successful for high Ca and low B but it would not be reasonable to assume a susceptibility index of zero. Increased susceptibility occurred for high N, K, Mg and Zn and for low Ca. Other treatment responses were not significant.

These data suggest that high N, K, Mg and certain other nutritional environments may increase susceptibility to fireblight. On equal importance, was the observation that low N did not decrease susceptibility. The decrease in susceptibility resulting from low P cannot be accepted as a practical approach because of difficulty of accomplishing this under field conditions plus the observations that this low level of P essentially eliminated flowering. The effect of high K on susceptibility need not be of any great concern because the control trees had an adequate level of K and the high level of K has not been found under field conditions. If higher levels of Mg do have a definite effect on susceptibility, this becomes a factor of reality in that the high Mg treatment resulted in an Mg level still slightly below the standard or diagnostic value in use. It may be desirable to use a lower Mg value for diagnostic work.

Table 2 presents the variation for certain specific Amino acids as associated with treatments or nutrient levels. As may be seen the nutritional environment had a definite influence on specific amino acids as well as total (Table 1). However, no specific relationship between susceptibility and total or a specific amino acid was apparent.

Table 2. Amino acids in Young Bartlett pear shoots as affected by nutritional status.

Nutrient Level	Amino acid - micrograms/100 mg fresh wt.*				
	Aspartic	Glutamic	Asparagine	Glutamine	Serine
Control	10	12	58	2	8
0.2 N	10	14	4	2	8
3 N	26	26	72	22	22
- P	12	10	60	2	6
5 P	14	22	94	18	18
- K	10	10	64	2	8
5 K	60	28	42	32	20
- Ca	10	14	32	2	8
5 Ca	10	26	38	6	16
- Mg	12	10	6	4	8
5 Mg	10	22	40	4	10
- Fe	12	10	30	2	4
15 Fe	8	20	26	14	8
- Zn	14	18	44	4	10
15 Zn	8	12	54	4	10
- B	10	12	26	4	10
15 B	4	8	6	4	8
- Mn	14	14	40	10	8
25 Mn	10	14	16	6	10
- Cu	12	20	28	6	10
25 Cu	10	20	32	10	12

* Other acids measured: Glycine, Threonine, Alanine, and Arginine.

The next step was to study the growth of Erwinia amylovora cultures to determine which nitrogenous compounds could be used by the organism as a sole source of nitrogen. The tests showed that Erwinia grew well when aspartic acid, asparagine, glutamic acid, glutamine, alanyl asparagin, glycine amide or ammonia (as NH_4Cl) was the only source of nitrogen. The bacteria did not grow well on 24 other amino acids.

The amino acids or soluble nitrogen compounds on which the bacteria grew had been shown to constitute about 80% of the total soluble nitrogen in pear shoots regardless of nutritional treatment. This leads to the conclusion that there is no nutritional control of fireblight.

Some reports suggest maintaining a lower level of K in pear orchards. This is a realistic approach in that applications of K fertilizers can be avoided until the tree is closer to a deficiency level. Sometimes, specific nitrogen carriers are suggested - i.e. use of nitrate nitrogen rather than ammonical forms. Research in Michigan has shown that this may have little or no effect. A five year study failed to show any differences between 6 sources of N and fall vs spring applications. The only difference found was as regards amount applied. When the N level got low, fruit quality was poor.

Some observations on general tree physiology appear to be in order. Common reports strongly suggest that "second" bloom and sucker (or water sprout) growth are of considerable importance in fireblight control. Sucker or water sprout growth becomes a real factor especially, when there has been a sizeable increase in nitrogen applications to older trees. These shoots continue to grow longer into the season than normal

terminal growth and, thus, provide a good source for late season infections. Secondary or "second" bloom has been referred to more often than I have seen it. However, we need to recognize that a second bloom can occur, especially if the tree has been through a period of stress that resulted in defoliation and/or early cessation of terminal growth.

Dr. Klos has observed recently that inoculations were essentially unsuccessful after terminal buds had set. His preliminary trials with Alar suggests that Alar, as a means of stopping terminal growth, reduces the success of inoculations. This is something that needs continued study. However, we need to be aware of the observation that stopping terminal growth early reduces production potentials and that Alar has been reported to reduce fruit size if applied within 2-3 weeks of bloom. Both reactions would be undesirable. However, the possibility of aiding in the control of fireblight with Alar or some other biologically active compound is extremely interesting.

To summarize - Present information indicates there is no nutritional control of fireblight. Some assistance may be obtained from certain biologically active compounds but, mainly, the horticulturist and pear grower will have to rely on control measures (sanitation and bacteriacides) advanced by the pathologists. Plant breeders may come to our rescue but this will not resolve the present problem soon enough.

Recovery of Erwinia amylovora from Apparently "Healthy" Pear Tissue

H. L. Keil[1/]

For a long time we have wondered what happens to Erwinia amylovora bacteria after a tree becomes infected but does not die. In order to resolve this question we set up the following study. Bartlett pear trees growing in 8-inch pots under greenhouse conditions were used. The plants were 2.5 to 3 feet tall and usually consisted of only one main shoot or whip. The plants were inoculated by hypodermic needle in the tip inch of the shoot. Blight infection took place and was allowed to run. In most plants, 8-12 inches of the shoot dies and then necrosis stops. After necrosis stops, one or two axillary buds below the necrotic area develop and grow into new shoots. Blight infection never develops in these shoots even though they are tender and succulent. Do these young tissues contain E. amylovora bacteria? We have, on many occasions, recovered E. amylovora from apparently "healthy" tissue below the dead tip and from the new shoot. The apparently healthy tissue, after surface sterilization with 10% clorox, was cut into 1 to 2 inch pieces. A 1/8 to 1/16 inch piece from the end of each 1 to 2 inch piece was placed on a nutrient yeast glucose agar (NYGA) plate and then incubated for several days. At the end of this time bacterial colonies developing from these pieces were transferred to NYGA slants and grown until sufficient material was available for pathogenicity tests. Bacteria from the slants were injected by hypodermic needle into the tips of tender succulent shoots of either Jonathan apple or Bartlett

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pear shoots growing in pots under greenhouse conditions. Bacterial colonies from apparently "healthy" wood having visible characteristics of E. amylovora proved to be pathogenic. In fact, pathogenic E. amylovora was isolated from "healthy" tissues after the plants were grown in the greenhouse for 6 months (May - October). These results indicate that E. amylovora may be translocated from disease tissue into apparently "healthy" tissue and remains viable in these tissues for long periods of time. Studies are planned to trigger visible symptoms by these organisms.

Can Fire Blight Resistance be Influenced by Amending Soils with Chemicals?

H. L. Keil [1]/

Several years ago while studying comparative fire blight resistance of susceptible and resistant pears, it was demonstrated that leaf ash inhibited growth of Erwinia amylovora. Certain chemical soil amendments of potted pear trees under greenhouse conditions apparently influenced size of inhibition zones produced by ash from young tender leaves which are vulnerable to fire blight infection. Among those compounds tested on the Bartlett variety, aluminum sulfate produced the greatest consistent increase in zone sizes. Spectographic analysis of leaves from plants growing in the amended soil showed no difference from plants growing in nonamended soils when analyzed for aluminum, boron, calcium, copper, iron, magnesium, manganese, phosphorus, sodium, and zinc. However, when analyzed for potassium, there appeared to be a positive correlation with E. amylovora inhibition. On a dry weight basis it appeared that if the potassium level of young tender leaves is below 1 percent, the inhibition zones are materially affected. It seems doubtful that the biological activity is due to the presence of a given concentration of potassium ion. In fact, studies conducted with potassium chloride and potassium sulfate at higher potassium ion concentrations than those found in leaves demonstrated no E. amylovora inhibition. We suspect that potassium is associated in some way with certain unidentified complex compounds responsible for resistance.

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One might suspect that the addition of aluminum sulfate makes the soil more acid which in turn allows roots to pick up more potassium. However, sulfur lowers soil pH more than aluminum sulfate but plants growing in soil amended with the latter material contain more potassium and generally show greater biological activity.

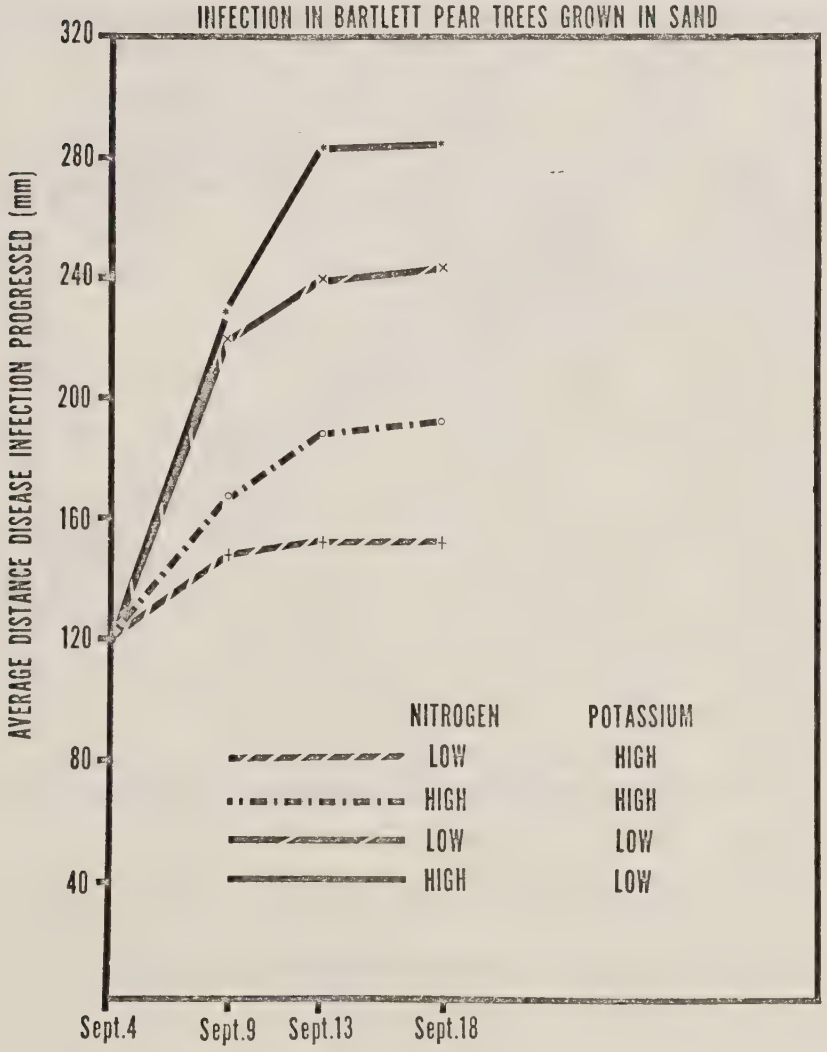
Correlation between plant potassium level and fire blight resistance appears to be supported by data documented by New York investigators. Two of these investigators, Drs. Parker and Luepschen, are here today and undoubtedly will want to make some comment during the discussion period.

Field studies are currently under way at Beltsville to see if the resistance of susceptible Bartlett pear can be improved by amending soil with various chemicals. In these studies sufficient nitrogen and phosphorus is supplied to produce maximum yield. Under these conditions the soil is amended with muriate of potash (300 lb/acre), aluminum sulfate (1,000 lb/acre), and ground lime stone (4,000 lb/acre). We expect this to be a long term study for at least 5 years unless fire blight destroys the trees before that time. Whether or not these trees will show enough resistance to reduce or overcome natural blight infection remains to be seen.

During the 1968 season Bartlett pear trees growing in sand in 5-gallon pails were amended with combination of high and low levels of nitrogen and potassium and optimum levels of other macro and minor elements. The trees were watered with nitrogen-potassium solutions usually twice each week. Even at the low levels of nitrogen (56 ppm) or potassium (19 ppm) the plants at no time showed any visible deficiency symptoms. August 23 ten plants of each treatment were inoculated by

hypodermic needle with 1/16 ml of E. amylovora suspension in the tip of the shoot. Measurement of disease progress in the shoots was made periodically after inoculation. Results are shown in the attached figure. This figure shows the least development of infection by low nitrogen and high potassium (100 ppm) and second lowest development by high nitrogen (225 ppm) plus high potassium. The greatest disease development was in plants growing in sand amended with high nitrogen plus low potassium and second highest disease development with low nitrogen and low potassium.

EFFECT OF NITROGEN AND POTASSIUM AMENDMENTS ON FIRE BLIGHT INFECTION IN BARTLETT PEAR TREES GROWN IN SAND





Dates of earliest known observations and secondary occurrences of fire blight in northern Europe

Role of Host Injury and Longevity of *Erwinia amylovora* in Development and Control of Fire Blight

T. van der Zwet [1/]

The role of host injury in the development of fire blight has been the object of much observation and experimentation. The length of time that *Erwinia amylovora* may live under different conditions has also received considerable attention. A series of experiments have recently been completed to study the role of injury and the longevity of *E. amylovora* in the development of fire blight and in its relation to control with streptomycin.

Materials and Methods

For all the experiments, we used Bartlett pear trees in 20 cm clay pots grown in the greenhouse to a height of 45-60 cm. In a preliminary test, sand, fine quartz gravel, and a large coarse gravel were compared with pyrax (pyrophyllite) at distances of 30 and 90 cm for injury of the pear tissues. Based on the degree of damage and on the amount of blight spread in the tissues, it was concluded to apply the fine sterile gravel from a distance of 30 cm at 70 psi in all experiments.

Inoculations were done by spray application of an aqueous cell suspension of *E. amylovora* at 70 psi. The suspension contained approximately 5×10^7 cells/ml, standardized to 70 units on a Klett-Summerson colorimeter with a no. 44 blue filter. Only the top 30 cm of the trees were used in the tests. All trees were incubated for 5 days in a lighted moist chamber maintained at 23°C and 100% relative humidity.

1/ Research Plant Pathologist, Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture, Beltsville, Maryland

In the first experiment, the Belair strain (pear) from Maryland was compared with strain 39 (apple) from Missouri. All 72 trees were spray-inoculated at the start of the experiment after which the gravel was applied immediately and at intervals of 1, 2, 4, 8, 16, 32, and 64 days after inoculation. Blight measurements were made 14 days after inoculation. In the second experiment, all trees were injured with gravel and then spray-inoculated with the Missouri isolate at intervals of 0, 1½, and 6 hours, 1, 2, 4, and 8 days after injury. One set of trees was incubated in the moist chamber and the other set remained on the greenhouse bench. Blight measurements were made 7 days after inoculation. In the last experiment, the trees were injured and spray-inoculated at the same time. They were then sprayed with 100 ppm streptomycin at intervals of 0, 1½ and 6 hours, 1, 2, 4, and 8 days after inoculation.

Results and Conclusions

In the test comparing the virulence and longevity of the Missouri and Maryland isolates, 24 trees inoculated with the Missouri isolate blighted versus only 12 trees with the Maryland isolate, regardless of the interval between inoculation and tree injury (Table 1). Trees sprayed with the Missouri isolate blighted faster than those sprayed with the Maryland isolate. Fdlowing injury at 4, 8, and 16 days after inoculation, all 12 trees sprayed with the Missouri isolate blighted, but only 2 trees (4 and 8 day interval) sprayed with the Maryland isolate. No blight appeared with either isolate after injury at 32 and 64 days after inoculation.

After cold storage for several months of all non-blighted trees, 3 trees originally inoculated (1 at 16, and 2 at 32 day intervals) with the Maryland isolate blighted when re-injured and incubated.

When all the trees were first injured and then inoculated with the virulent Missouri isolate, entry and spread of blight was faster when trees remaining on the bench were inoculated within 1½ hours after injury than those inoculated 1-8 days later (Table 2). This also seemed apparent for trees incubated in the moist chamber and inoculated within 6 hours after injury than those inoculated 1-8 days later. Trees inoculated 2-8 days after injury and allowed to remain on the bench showed fewer infected trees than those inoculated at shorter intervals. All the trees placed in the chamber became blighted regardless of the time of inoculation. There is no adequate explantation for the blighted tree in the gravel check. Infection may have occurred through splashing in the moist chamber.

When trees were inoculated with E. amylovora and were then sprayed with streptomycin at different intervals, complete control was obtained when uninjured trees were sprayed within 6 hours after inoculation (Table 3). Partial control was obtained when injured trees were sprayed within 1½ hours after inoculation. Fewer uninjured than injured trees blighted when streptomycin was applied 1-8 days. Blight infection progressed less in uninjured trees sprayed 1-8 days after inoculation than it did in injured trees.

It seems apparent that the Missouri isolate of E. amylovora is more virulent than the Maryland isolate. On the other hand, the Maryland isolate seemed to survive longer than the Missouri isolate. Trees inoculated within 24 hours following injury showed considerably more blight development

than trees inoculated from 1-8 days later. Fire blight was completely controlled with 100 ppm streptomycin when uninjured trees were sprayed within 6 hours following inoculation. Injured trees were partially controlled when they were sprayed within 1½ hours after inoculation.

Table 1. Fire blight development in Bartlett pear trees after injury at different intervals following inoculation with a Missouri or Maryland isolate of *Erwinia amylovora*.

Interval between inoculation and injury	Trees blighted		Length of Blighted Shoot[1/]			
			Maximum		Average	
	Mo.	Md.	Mo.	Md.	Mo.	Md.
days	number	number	cm.	cm.	cm.	cm.
0	4	4	33	30	29	25
1	4	4	38	29	30	21
2	4	3	35	46	32	23
4	4	1	55	34	30	9
8	4	1	64	23	54	6
16	4	0	46	0	40	0
32	0	0	0	0	0	0
64	0	0	0	0	0	0
checks						
Gravel	0	0	0	0	0	0
Bacterium	4	3	59	23	32	13

1/ 14 days after inoculation

Table 2. Fire blight development in Bartlett pear trees after inoculation at different intervals following tree injury.

Interval between injury and inoculation	Trees blighted		Length of Blighted Shoot 1/			
			Maximum		Average	
	Chamber	Bench	Chamber	Bench	Chamber	Bench
hours	number	number	cm.	cm.	cm.	cm.
0	4	4	33	35	27	22
1½	4	4	30	38	23	26
6	4	4	23	18	21	12
days						
1	4	4	25	11	18	9
2	4	2	21	18	15	7
4	4	4	18	19	10	11
8	4	3	13	8	8	3
checks						
Gravel	1	0	6	0	2	0
Bacterium	1	2	8	18	2	6

1/ 7 days after inoculation

Table 3. -- Effect of streptomycin sprays on control of fire blight in Bartlett pear trees, applied at different intervals after spray inoculation with Erwinia amylovora, with and without injury of the tree tissue.

Interval between inoculation and strep application	Trees blighted		Length of Blighted Shoot 1/			
			Maximum		Average	
	Injured	Uninjured	Injured	Uninjured	Injured	Uninjured
hours	number	number	cm.	cm.	cm.	cm.
0	2	0	25	0	7	0
1½	2	0	21	0	6	0
6	4	0	35	0	23	0
days						
1	4	1	40	33	25	8
2	4	1	34	35	26	9
4	4	3	40	41	33	29
8	4	2	31	30	28	14
Checks						
Gravel	--	--	0	--	0	--
Bacterium	--	2	--	38	--	13
Bacterium + gravel	4	--	43	--	34	--

1/ 14 days after inoculation

Influence of Pear Tissue Age on Infection by Erwinia amylovora

T. van der Zwet[1/]

Susceptibility to Erwinia amylovora varies considerably among pear varieties. Succulent shoots, 2-year-old branches and green fruit on 5-year-old field-grown trees of 11 varieties of Pyrus communis and clone #76 of P. ussuriensis were inoculated under optimum blight conditions. The inoculum consisted of a 0.25 cc aqueous cell suspension (30×10^7 cells/ml) of E. amylovora. The shoots and fruit were inoculated with a 22-gauge hypodermic needle, whereas the older branches were injected beneath the bark after previous wounding. Blight measurements were made 10 and 20 days after inoculation of all the tissues and, in addition, 80 days after inoculation of the older branches (Table 1).

All fruit blighted severely within 3 weeks following inoculation. On the basis of the degree of blight development in the stem tissues, the varieties were divided in three general groups:

1) Varieties extremely susceptible in both the young shoots and in 2-year-old branches,
2) Varieties moderately susceptible in the young shoots but resistant in the older branches, and
3) Varieties moderately to highly resistant in the young shoots but extremely susceptible in the older branches.

In the blight susceptible varieties (Bartlett, Stewart Bartlett and Dawn) the disease spread rapidly in both the young and old branch tissues. In

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Table 1. Maximum and average blight development in succulent shoots, 2-year-old branches and green fruit of 11 varieties of Pyrus communis and a clonal selection of P. ussuriensis after artificial inoculation under field conditions.

Pear Variety	Shoot Blight 20 days after inoc. max.	Shoot Blight 20 days after inoc. avg.	Branch Blight 80 days after inoc. max.	Branch Blight 80 days after inoc. avg.	Fruit Blight 1/ 10 days after score	Fruit Blight 1/ 20 days after score
	cm.	cm.	cm.	cm.		
St. Bartlett	38	23	190	120	++	++++
Bartlett	53	45	138	80	+	++++
Dawn	43	25	80	20	no fruit	
R.C.W.	53	10	0	0	+	++++
Rich. Peters	18	2	0	0	+	++++
Kieffer	10	8	0	0	++	++++
Waite	6	6	0	0	+	+++
Old Home	5	1	0	0	+	++++
Maxine	23	20	13	5	0	++++
Magness	6	3	70	28	0	+++
Farmingdale	38	3	120	45	+	+++
P. ussur. 76	0	0	115	28	0	++++

1/ Fruit blight rating: 0 - none; + - light; ++ - medium; +++ - severe; ++++ - very severe

6 varieties, fire blight was moderately severe in the young shoots, but the 2-year-old branches seemed highly resistant to the development and spread of fire blight. In the resistant Magness variety and in P. ussuriensis 76, the young shoots were highly resistant to fire blight. In Farmingdale, the young shoots seemed moderately resistant. In the older branches of two of the trees of each variety and the selection, however, the disease spread beyond expectation into the central leader and throughout the tree. Young shoots of these trees may possess some resistance factor which may be absent or unoperative in the older branches.

Incidence of Twig and Fruit Blight on 4 Varieties of P. communis

T. van der Zwet and H. L. Keil[1/]

During the late spring and early summer of 1968, weather conditions were extremely favorable for fire blight development in the pear plantings at Beltsville, Maryland. Several severe rain storms, once accompanied by light hail, struck the pear variety orchard. In early July, first occurrences of fruit blight, without twig infection, were observed in the Devoe and Moonglow varieties. Symptoms of twig blight without the presence of fruit blight were observed.in the Bartlett and Stewart Bartlett varieties. Counts were made on July 17 and July 31 of both types of blight on 16 trees each of the four varieties. The final count is tabulated below.

Blight Incidence	Bartlett		Stewart Bartlett		Devoe		Moonglow	
	no.	%	no.	%	no.	%	no.	%
Trees with twig blight	0		3		0		2	
Trees with fruit blight	0		2		3		6	
Trees with both blights	16		10		12		2	
Trees without blight	0		1		1		6	
Trees total in row	16		16		16		16	
Trees blighted		100		94		94		63
Twigs blighted	262		201		144		7	
Fruit on all trees	753		368		2461		868	
Fruit blighted		19		15		31		9

The two Bartlett varieties averaged 1.6 times more twig blight than Devoe, whereas the latter had 1.8 times more fruit blight. Artificial inoculation of the fruit of all 4 varieties proved negative in uninjured fruit but highly successful in injured fruit.

1/ Research Plant Pathologists, USDA, Fruit and Nut Crops Research Branch, Plant Industry Station, Beltsville, Maryland

Table 1. Results of testing Pyrus selections for resistance to fireblight.

H. R. Cameron[1/]

Species or Cultivar	Source of Material	Type of Source a/	Tree b/ Infection	Shoot c/ Infectio (Average %)
P. amygdaliformis Vill.	Greece, (Epires, Macedonia)	Seedlings(3)	13/15	43
P. amygdaliformis Vill.	Turkey	Seedlings(4)	15/15	48
P. betulaefolia Bunge	China	Seedlings(2)	0/20	00
P. betulaefolia Bunge	China	Clone(4)	0/19	00
P. betulaefolia Bunge	Italy	Seedlings(1)	10/10	80
P. calleryana Decne.	U. S. Nurseries	Seedlings(3)	22/37	24
P. calleryana Decne.	China	Clone(3)	0/17	00
P. calleryana Decne.	China	Clone(1)	2/3	75
P. calleryana Decne.	Oregon	Clone(4)	0/14	00
P. calleryana Decne.	Oregon	Clone(8)	11/31	7
P. communis L. domestica				
Bartlett 0 P-9	Oregon	Clone(1)	12/13	71
Bartlett A	California	Clone(1)	4/5	20
Bartlett B	California	Clone(1)	5/5	56
Bartlett C	California	Clone(1)	4/5	20
Bartlett D	California	Clone(1)	4/5	36
Bartlett Stewart	Washington	Clone(1)	4/5	52
Eldorado	California	Clone(1)	6/6	37
Old Home	Oregon	Clone(1)	0/15	00
Old Home x Farmingdale	Oregon	Clone(64)	343/457	20
Old Home x Farmingdale	Oregon	Clone(24)	0/114	00

1/ Plant Pathologist, Oregon State University, Corvallis, Oregon

P. communis L.	France	Seedlings(1)	16/18	62
P. communis L. (wild)	Russia	Clone(4)	4/4	60
P. communis L. (wild)	Russia	Clone(2)	0/2	00
P. communis L. (wild)	Turkey	Seedlings(6)	33/34	85
P. elaeagrifolia Pall.	Turkey	Seedlings(3)	17/22	27
P. Fauriei Schneid.	Korea	Seedlings(2)	1/10	2
P. Koehnei Schneid.	California	Clone(1)	6/8	87
P. pashia Ham.	N. India	Seedlings(1)	10/10	116
P. pashia Ham.	India	Seedlings(1)	3/8	23
P. pashia Ham.	W. Pakistan	Seedlings(1)	9/9	71
P. salicifolia	Illinois (Morton Arb.)	Clone(1)	2/5	13
P. syriaca Boiss.	Israel	Seedlings(1)	3/3	47
P. ussuriensis Max.	N. China	Clone(1)	0/5	00
Cydonia oblonga (Lepage series "C" Provence quince)	France	Clone(1)	2/5	12
(East Malling quince A)	England	Clone(1)	4/5	32

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a/ 5 trees of each clone were usually used. ( ) shows number of seed sources or of clones tested.

b/ number of trees infected/number of trees inoculated. 5 shoots inoculated per tree.

c/ blight infected shoots/number of inoculations made.

Table is from manuscript submitted for publication.

CAMERON, H.R., M. N. WESTWOOD and P. B. LOMBARD. 1969. Resistance of Pyrus species and cultivars to Erwinia amylovora (Burr.) Winslow et al.

Duane L. Coyier
U. S. Dept. of Agriculture
Hood River, Oregon

Inoculation of apple and pear roots with E. amylovora.

Infection of apple and pear roots with Erwinia amylovora via infection of the aerial portion of the plant is well known. The effect on the aerial portion of the plant following inoculation of the roots has received little attention. An investigation into the cause of poor growth of newly planted pear trees revealed the following facts:

1. Little or no growth of the above ground portion of the plant.

2. No cankers, ooze or other symptoms of fire blight were evident.

3. No development of feeder roots.

4. Bacteria were present and isolated from the root system of the Bartlett trees in the block (50 plants).

5. Re-inoculation of the bacteria into young pear fruitlets and pear seedlings produced typical fire blight symptoms.

It was concluded that the roots had been accidentally inoculated by fire blight bacteria during the planting operation through contaminated pruning tools.

An experiment was conducted to test this theory. Young pear and apple seedlings were used as test plants and all were removed from the soil. The roots were washed to remove all soil particles. Half the plants were root pruned with sterile tools and replanted in gallon cans. The other half were inoculated with various virulent cultures of E. amylovora via pruning tools contaminated with bacteria from isolations mentioned previously.

Results of the test showed a marked difference in growth between inoculated and non-inoculated plants. Many of the plants inoculated with E. amylovora died following a prolonged period of poor growth, Others did not succumb to the infection, but neither did they attain growth comparable to the check plants.

We concluded that E. amylovora was responsible for poor growth of the pear trees although no typical symptoms of fire blight were

observed. Since similar poor growth of pear seedlings is a frequent problem in commercial orchards we advised using caution in pruning and handling of young trees during the planting process.

Control of Fire Blight with Bacticin.

Bacticin (2,4-Xylenol-0.463% w/w + meta-cresol-0.466% w/w) was developed by the Upjohn Co. for treatment of crown gall on various fruit and ornamental trees. Since it has some degree of systemic activity we tested it for control of fire blight cankers on pear trees. We inoculated susceptible young shoots of Forelle pear trees (container grown in the greenhouse) and painted the shoot with full-strength Bacticin at a point below the inoculation site. Similar trees were inoculated without Bacticin treatment to serve as checks.

The bacteria spread rapidly downward killing the tissue and producing typical bacterial ooze in untreated plants. Fire blight also developed near the inoculation site of treated plants, but did not move through the Bacticin-treated area for 3-4 weeks following inoculation. Our observations would lead us to believe that Bacticin penetrated the bark and acted as a bacteriastatic agent. As it becomes diluted or degraded in the plant the bacteria move through the treated zone to invade the plant in a typical manner.

The work reported here is preliminary and further investigation is planned which will be supported by an Upjohn Co. research grant.

Another phenomenon of interest was accidentally discovered while treating fire blight cankers on Bartlett pear trees. Following removal of the infected wood we treated the general area with Bacticin and found that in some cases we had not removed all the infected area. Although there was no visible evidence of infection, areas invaded by the bacteria turned black within a few minutes after treatment with Bacticin. Further investigation revealed that this reaction apparently does not occur to the same extent with all varieties and under all conditions. Further studies will be undertaken to determine the factors involved in this rather unusual phenomenon.

Various Aspects of Fireblight Control

Edward J. Klos

Dept. of Botany & Plant Pathology

Michigan State University

INTRODUCTION

In 1954, John C. Dunnigan wrote in the Agricultural and Food Chemistry the following, "In the Eastern U.S. pear blight is a limiting factor in production of high-quality pears...Even in the Far West where the climate is less humid pear blight is a constant threat..."

He goes on talking about copper sprays and their disadvantage of fruit russetting. He mentioned development of Streptomycin and the exceptional control during bloom.

Today in 1969 these statements comply to not only pear blight but apple blight. I believe most pathologists will agree that streptomycin used properly during bloom will control blight at this time. However, our weakest part of our control program is during active twig growth period.

In the past fifteen years no new effective chemical control has been developed for use against fireblight.

The U.S.D.A. breeding program produced two selections namely Magnus and Moonglo, Purdue and Ohio State are other areas where breeding research is conducted in this country for fireblight resistance.

In recent years with the spread of fireblight in Europe and the increase of its incidence in many Eastern states on both apple and pear this disease has been put into focus with fruit pathologists, horticulturists, and entomologists.

At Michigan State University a new project was initiated in 1968 titled, "Fireblight, Its Etiology and Control". Three departments are co-operating, Botany & Plant Pathology, Entomology and Horticulture.

PERSONNEL: E.J. Klos (Bot. & Plant Path.) (Project Leader), A. Kenworthy R. Carlson (Hort.), and J. Bath (Ent.).

OBJECTIVES:

A. Pathological

1. To develop a more economical fireblight control program during bloom and/or during the post bloom infection periods. Emphasis will be given to developing systemic compounds to eliminate costly eradication of cankers and other infected tree parts.

2. Expand the research program in determining the role of yellow bacteria in the life cycle of the fireblight organism.

3. To study etiology of the organism.

B. Horticultural

1. Change the physiology of the tree by use of fertilizers and biologically active chemicals to observe effects upon resistance to fireblight and interaction with pesticide application.

2. Survey for resistant individuals from susceptible varieties.

C. Entomological

1. Study the disease-vector relationships involved in the spread of fireblight with particular emphasis on aphids, plant bugs, leafhoppers and pollinating insects.

2. Develop insect vector chemical control methods to reduce spread and transmission of fireblight.

In Botany and Plant Pathology the following personnel are involved in fireblight research:

Dr. E. J. Klos--Advisor
Jon Robb--1/2 time technician, chemical control-laboratory greenhouse and orchard plots
John Riggle--Graduate Student--Role of yellow bacteria in life cycle of fireblight organism and ecology of both
Calvin Waisanen--Graduate Student--host-parasite relationship

1. Chemical Control

a. Compound UC17525 (Bromoacetyl valinamide) discovered to be a bacteriacide against the fireblight organism several years ago at M.S.U. during Dr. L. Lewis' (Hort.) Ph.D. research problem. This compound and its derivatives showed promise in orchard tests in Michigan and New York. Development of this compound was discouraged by the manufacturer.

In the past six years several hundred bacteriacidally active compounds were evaluated in our laboratory against the Erwinia amylovara. Promising compounds were further evaluated in the greenhouse and field.

In addition a number of fungicides were evaluated alone or in combination against the organism in vitro. The following compounds--Difolatam,

Cyprex Captan, Difolatan + Cyprex, Difolatan + Captan, Cyprex + Captan all gave inhibition zones slightly less than Streptomycin. Dupont 1991 showed no activity.

In Table I is a summary of part of our laboratory study of the most promising compound as compared to streptomycin sulfate.

Table I. Zone Inhibition

		Exp. I-4	Exp. I-5	Exp. I-8
Streptomycin	100 ppm	2.9*	2.9	2.2
LMA-B-100	1000 ppm	4.3	--	--
LMA-B-100	100 ppm	3.3	2.7	2.2
LMA-B-100	50 ppm	--	--	N.I.

*Centimeters

N.I. = Negligible Inhibition

LMA-B-100 is a antibiotic produced by Abbott under the name of Spectinomycin. At 100 ppm it was comparable to Streptomycin at the same concentration.

This compound and a number of others were tested in the greenhouse against twig infection.

Table 2. Greenhouse Experiments - Fireblight Chemical Control.

April 25th sprayed with bactericide then prick inoculated. Counts 5-10
(Variety Jonathan)

Compound	Rate	% Infectivity (Mean)
TD235	1000 ppm	81.0
TD439	1000 ppm	69.0
TD5177	1000 ppm	63.0
E11241	1000 ppm	88.0
LMA-B-100	1000 ppm	25.0
Agri-Mycin (Streptomycin)	100 ppm	19.0
D.W. (check)	--------	100.0

In Table 2, LMA-B-100 gave comparable results to Agri-Mycin (Streptomycin) in greenhouse twig infection on Jonathans. The remainder of the compounds showed no significant control over the checks.

Table 3. Orchard Experiments - Fireblight

Jonathan Twigs

Sprayed July 1 with bacteriacide, prick inoculated July 2. Counts 7-22

Compound	Rate	Length of Infectivity (Mean)
Agri-Mycin	100 ppm	6.6 inches
LMA-B-100	1000 ppm	5.4 inches
TD5177	1000 ppm	14.5 inches
TD439	1000 ppm	14.2 inches
ELI241	1000 ppm	11.2 inches
Check	--------	16.5 inches

In Table 3, the compounds were applied one day before inoculation. In this experiment, LMA-B-100 at 1000 ppm was comparable to streptomycin.

Table 4. Orchard Experiments - Fireblight

Jonathan Twigs

July 25th sprayed twig (except first five leaves) with bacteriacide, prick inoculated at tip on July 27th. Counts 8-18.

Compound	Rate	Length of Infectivity (Mean)
LMA-B-100	1000 ppm	15.6 inches
LMA-B-100 Surf	1000 ppm & .35 ml/L	13.1
LMA-B-100 & 1991	1000 ppm & .56 gr/L	18.0
LMA-B-100	3000 ppm	3.1
LMA-B-100	5000 ppm	14.6
Strep	100 ppm	12.6
LMA-B-101	1000 ppm	6.8
LMA-B-101	5000 ppm	1.3
Check	--------	19.8

In Table 4 the results indicate that there is redistribution or systemic action of LMA-B-100 & LMA-B-101. The various rates of LMA-B-100 gave varying degrees of control. However, the above experiments indicate that these compounds show promise as bacteriacides for fireblight.

Table 5. Fireblight Chemical Control Research - Summary

Material	Supplier	Lab	Potential G. House	Field
Agri-Mycin	Pfizer	+	+	+
LLL-B-100	Amdel	+	+	+
LLL-B-101	Amdel	?	?	+
C-54	Uni Royal	+	-	?
TB113	Pennsalt	+	+	?
TB439	Pennsalt	+	+	+
TB5177	Pennsalt	+	+	-
LLL241	Eli Lilly & Co.	-	+	+

+ = Active - = No activity

Table 5 is a summary of the more promising compounds as to their potential for fireblight research namely LMA-B-100, LMA-B-101, TD439.

2. Yellow Bacteria

In the past 3 years several experiments were conducted to study the antagonism of yellow bacteria and Erwinia amylovora. Rosen, Farabee, and Lockwood and Goodman have studied this antagonism especially with twig infection.

In 1966 an experiment was set up to study the antagonism of yellow and fireblight bacteria when grown together at various intervals. In table 6 is an experiment where the period of 48 hours was used for the growth period.

Table 6. Infectivity Experiment with Yellow and Fireblight Bacteria

Treatment	Number of Terminals Infected*	Mean length of Infected Tissue	% Infected
Yellow + White (48 hrs.) (Incubated together)	2	2.1 inches	16.7
Yellow and White (Incubated separately 48 hrs.) Combined before inoculation	8	9.0	66.7
White alone Incubated 48 hrs.	12	12.9	100.0
Yellow alone incubated 48 hrs.	0	0.0	0
Prick (Check)	0	0.0	0

*Twelve randomized Jonathan twigs used in each treatment

This experiment shows that influence of yellow bacteria on the fireblight bacteria infectivity was similar under controlled orchard conditions as in the greenhouse experiments. That is the longer the two bacteria are together up to 48 hours, the greater the reduction in infectivity.

In 1968 an experiment was conducted during bloom in which yellow bacteria were sprayed in and on the blossoms 24 hours before inoculating them with Erwinia amylovora.

Table 7. Yellow Bacteria Antagonism in Bloom Against Erwinia Amylovora.

Bartlett

M.S.U. Orchard

Treatment	% Infected	Severity 0-5
Yellow followed by Fireblight Bacteria 24 hours later.	20	0.25
Fireblight Bacteria alone	40	1.40
Yellow Bacteria alone	0.0	0.0

In Table 7, it is shown that the yellow bacteria are antagonistic against the white by reducing the percentage of infected bloom from 40 to 20.

In addition to the above antagonistic work an ecological study of the bacteria is being conducted. The results are too preliminary to make any statements at this time.

3. Alar Experiments (Cooperator Dr. A. Kenworthy).

As stated in the introduction the chief weakness in control of fire-blight is the twig stage. It occurred to us that if growth retardants could be applied to stop growth the incidence of twig blight could be greatly reduced during the summer.

Table 8. Effect of Alar on Susceptibility of Bartlett Pear to Fireblight Injection.

Treatment	% Infected (Terminals)
1. Alar 1000 ppm	23.3
2. Check	60.0

In Table 8 the terminals were treated on June 5 and inoculated with bacteria on June 16 and readings were made on July 19. The terminal bud was set within 21 days in this experiment in most cases. This setting of the bud was also reflected by the number of infected terminals as compared to the check. This treatment shows promise.

Table 9. Alar Concentrations Study Against Fireblight Infection

Variety - Bartlett

Treatment	% Infected	Inches Infected (Mean)
Alar 1000 ppm	17	.6
Alar 2000 ppm	25	1.1
Alar 5000 ppm	25	.6
Check	42	1.8

In this experiment is the different concentrations of Alar used showed no significant difference in infection. Rates of 1000 ppm or less will be tested in 1969.

This method of control should be further investigated as to time of application concentration and effects on tree and yield.

[illegible] - [illegible]

[illegible] - parasite relationships studies were initiated last fall by [illegible] by Mr. J. Heisman. The objective of this study is to determine the [illegible] and method of infection by the bacteria and the effects on the host (pear). This information will be useful in understanding the process of infection and aid in developing a control practice.



I'LL BE DARNED!!

SUMMARY OF FACTS ON ERWINIA AMYLOVORA FROM THE LITERATURE

Bacteriology

Gossard 1916	E. amylovora remains virulent in honey for 47 hours.	(III-4)
Pittman 1929	E. amylovora destroys 8.2% of total pectin in apple at room temperature (aerobically).	(III-11)
Shaw 1929	E. amylovora is killed by yellow organism in 4-8 hours in nutrient dextrose broth and in blighted tissue.	(M.S.,Ark.)
Pierstorff 1931	E. amylovora can live in acid media of pH 4.6, can withstand -183°C for 10 minutes, and heating to 48°C does not always inhibit growth.	(III-12)
Ark 1940	Duration of life of E. amylovora prolonged by addition to potato dextrose agar of vitamin C, cystine, glutathione, pyrogallol, resorcinol, and tannic acid.	(III-31)
Ark 1941	Bacteriocidal substances against E. amylovora produced by Bacillus vulgatus and yellow bacterium isolated from soil; substances are water soluble, were able to withstand boiling for 60 minutes, and were active in extremely small quantities.	(III-37)
Elrod 1941	E. amylovora serologically homogeneous.	(III-35)
Kent 1942	E. amylovora can utilize only complex organic sources of nitrogen.	(III-40)
Starr 1950	All 12 strains of E. amylovora required 10 μgm nicotinic acid/100 ml NH_4Cl glucose and salts basal medium for optimum growth.	(III-47)
Hildebrand 1954	Shortest bacterial cells with most and longest flagella make up the most virulent cultures. Generation time of E. amylovora ranges from 55-155 minutes: avirulent cultures 72-77 minutes; virulent cultures 80-82 minutes.	(III-49)
Reinhardt 1957	Anaerobically grown E. amylovora is more sensitive to freezing. Apple juice and bacterial ooze protect bacteria from death by freezing. E. amylovora grown aerobically in yeast extract broth decreases pathogenicity.	(III-62)
Sutton 1958	E. amylovora does not grow anaerobically in media with mineral salts, glucose and nicotinic acid. Cell suspensions, grown aerobically or anaerobically, are capable of anaerobic dissimilation of glucose in buffer.	(III-58a) (III-60)

Farabee 1958	Optimum pH of antagonistic bacteria is below pH 4.4 which inhibits E. amylovora in culture by lowering pH of medium favorable for growth.	(III-58)
Shaffer 1961	Rough form of E. amylovora is avirulent, reaches maximum growth in vitro after 11 hours and produces endo-toxic substance; smooth form is virulent, reaches maximum growth in 50 hours and produces an exo-toxin.	(III-66)
Lewis 1961	E. amylovora readily uses aspartic acid, glutamic, asparagine, glutamine, beta- alanine, gamma- amino butyric acid as a source of nitrogen in synthetic culture medium. Bromoacetyl valinamide kills E. amylovora.	(III-70)
Baldwin 1962	Yellow isolate probably aberrant form of E. amylovora; acid production in presence of sucrose; slow growth on media with salicin.	(IV-48)
Goodman 1962	The reversion of E. amylovora from the avirulent rough to the virulent smooth form on exposure to aphid extracts.	(III-63)
Goodman 1965	A white virulent form (35-W) of E. amylovora and yellow avirulent form (35-Y) grew at the same rate in vitro; the longer they were incubated together before inoculation into Jonathan tree, the less infectious they became.	(III-76)
Powell 1965	Observed direct relationship between inoculum concentration, length of incubation and amound of infection: 200 cells/ml, no infection; $20x10^3$ cells/ml, 40% of inoculated shoots developed blight after incubation of 7-10 days; $20x10^6$ cells/ml, 100% infected shoots after incubation of 4-5 days.	(VI-15a)
Starr 1966	Bdellovibrio bacteriovorus, a predatory ectoparasitic and bacteriolytic microorganism on E. amylovora.	(III-69b)
Smith 1966	Yellow isolate obtained from a Jonathan tree canker was non pathogenic to Jonathan. Electrophoretic soluble protein patterns differed between this isolate and E. amylovora.	(III-83a) (III-88)
Goodman 1967	Yellow avirulent bacterium ($5x10^6$ cells/ml) provided protection against infection by virulent strain of E. amylovora ($5x10^4$ cells/ml) when inoculated 30 minutes later	(III-84)
Burkowicz 1968	Generation time of virulent isolate 3.96 hours, avirulent 3.74 hours; no necrosis with E. amylovora inoculum containing less than 10^5 cells/ml.	(Ph.D,Mo.)

ETIOLOGY AND EPIDEMOLOGY

Hotson 1916	Longevity of E. amylovora on blighted branches: exposed to direct sunlight, stay alive 10-13 days; part sunlight part shade, stay alive 27 days; under cover crops, stay alive 29 days.	(IV-5)
Brooks 1926	Alternate cloudy and sunny periods favor canker exudation. Exposure of ooze to direct sunlight killed bacteria in 22 hours. Rain caused 90% of twig infection in 1925.	(VI-3)
Nixon 1927	Observed 2 stages in life cycle of E. amylovora: a vegetative stage in which zoogloea migrate intercellulary forming schizogenous cavities and a dormant or pseudo fructification stage in which bacterial cysts are formed intracellulary in lysigenous cavities.	(IV-12)
Tullis 1929	Rain plays considerable part in fire blight from primary sources of infection. Stomatal infection very important in development of twig blight.	(IV-16)
Rosen 1929	Bacteria move intercellularly, dissolving cell walls, causing plasmolosis; bacteria never observed in the protoplast; bacteria were observed in the phloem, xylem, and pith.	(IV-36)
Rosen 1933	Ooze from infected apple trees did not contain viable bacteria. Could not establish correlation between amount of blight from one year to the next. Suggested 4 ways of primary infection: 1) internal extension of last year's blight; 2) bud infection in the previous year; 3) infection from last year's ooze; 4) twig blight caused by live cankers.	(IV-37)
Pierstorff 1934	Carefully conducted experiments showed the bees carry bacteria from artifically inoculated apple blossoms to other blossoms on the same tree; bees did not cause infection when inoculated hives were placed under caged apple trees. Longevity of E. amylovora was 24 hours in combs and frames, 2 days inside bees, and 5-11 days in pure honey.	(V-22)
Thomas 1934	Growth of E. amylovora in culture solution is optimum at 10% or less of sugars; growth is inhibited at 18-20% of sugars, Nectar of pear blossoms contain in dry weather 50% sugar, in humid weather about 2%.	(VI-5)
Rosen 1936	In artifical inoculation of pear flowers, bacteria were observed under the nectarial tissue within 48 hours; on apple flowers, penetrations occur more frequently through stigmas, anthers, and calyx lobes.	(IV-40)

Parker 1936	Sucking insects appear of importance in primary and secondary infection of terminal shoots; rain and insects are responsible for leaf infection.	(IV-41)
Ark 1936	E. amylovora was isolated from internal organs of pupal and adult stage of Drosophilia melanogaster and Musca domestica and externally from eggs of M. domestica.	(V-26)
Rosen 1938	Bacterial exudate contains viable bacteria when kept at 16°C and humidity of 0-45%; also at temperatures of 25-40°C and low humidity but died at 45% R. H. Exudate kept at fluctuating outdoor temperatures: at 0% R. H., all bacteria viable for 12 months; at 50% R. H., viable for 9 months; at 70-90% R. H., all dead. Bacteria in exudate contain slimy capsules; in pure cultures no capsule.	(III-27)
Ivanoff 1937	Aerial strands of E. amylovora measure 8-45 u in width and contain infectious bacteria.	(VI-10)
Ivanoff 1941	In nectar drops containing 20,30,40-50% sugars, bacteria survived for 48, 24, and less than 24 hours, respectively. In seeded test tubes filled with artificial nectar solution (40% sugar) enough bacteria survived for 72 hours to induce pear blossom infection. Blossom inoculation in the greenhouse with a 1/400 cc water drop with: 10 bacteria, caused 66% infection; 100 bacteria, 91%; and 1000 bacteria, 93%.	(V-30)
Hildebrand 1947	100 single cells of E. amylovora applied to pear blossoms gave no fire blight. On apple flowers in moist chambers at 24°C, positive results were obtained with: 9 out of 15 single cell inoculations, 3 out of 5 two-cell inoculations, 4 out of 5 five-cell inoculations, and all with 10-cell inoculations.	(IV-23)
Baldwin 1962	High percentage of apple buds yielded E. amylovora phage-sensitive isolates in mid-winter.	(IV-48)
Lewis 1965	Showed definitely that E. amylovora can enter Jonathan apple through upper leaf surface hydathodes, trichomes and stem lenticels; migration in the stem occurs through the phloem.	(IV-50)
Plurad 1965	Showed persistance of E. amylovora in the apple aphid.	(V-34)
Powell 1965	Determined minimum temperature necessary for blossom blight on basis of number of "degree days": 30 degree days above 65°F are needed between latest pre-bloom freeze and early bloom.	(VI-15a)

Bauske 1966	Fire blight spread in direction of wind; in wind protected positions 6% of trees blighted compared to 59% of trees in exposed areas. Aerial strands observed on infected stems and petioles, are readily airborne and are infective after dissolved in water. Strands infective after 1 year at 5°C. Chemical control of insects does not control fire blight	(IV-51)
Keil 1966	Showed longevity of E. amylovora on Bartlett trees in the greenhouse when the trees were sandblasted 4 weeks after spray inoculation.	(VI-17)
Burkowicz 1968	Permeability and ultrastructural changes in apple and pear tissue induced by E. amylovora.	(IV-53) (IV-54)
Wilson 1968	Increase in fire blight severity with sprinkler irrigation.	(IV-55)
van der Zwet 1969	Only 10% of cankers studied produced ooze and were located in 4-8-year-old wood, if blight had entered the tree through a side branch; all ooze was infectious; shifts in ratio between fire blight and other bacteria were noticed during the winter.	(III-87)

Shaw 1935

% R.H.	% I.H.	% Shoot blighted
100	100	100
70	99	30
	98	5
	97	0

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BLIGHTER'S ADVENTURES

DORMANT PERIOD



Frozen in sleep, perhaps the Blighter dreams of Spring



After the fast of Winter they are ready for the feast of Spring

EARLY BLOSSOM PERIOD

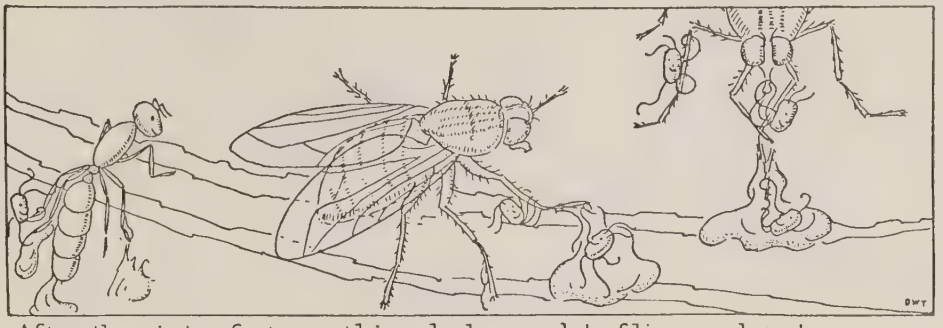


The Blighters indulge in an orgy of drinking

(AFTER HILDEBRAND, 1939)

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BLOSSOM PERIOD



After the winter fast, anything looks good to flies and ants



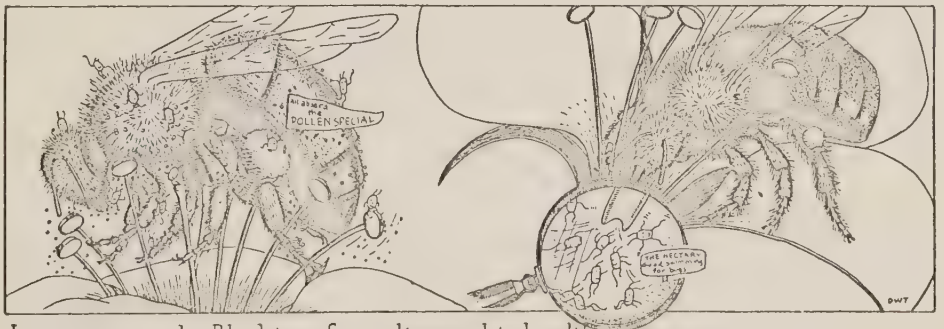
The fly sometimes relishes a change in diet



The Blighters are washed - from one place to another



Both pollen and nectar are parts of bee diet

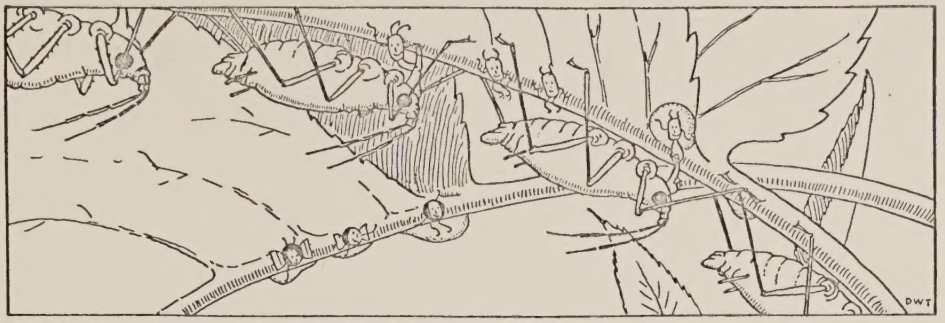


Insects carry the Blighters from diseased to healthy blossoms



The busy bee ranges far and free, and takes the Blighter with him

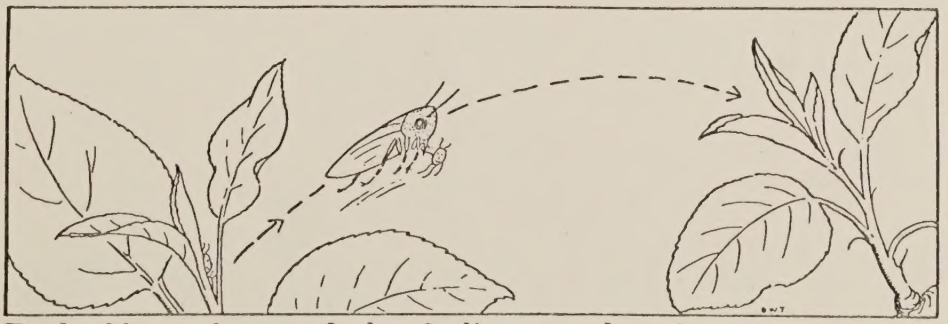
LATE SPRING AND SUMMER PERIOD



When aphids feed, the Blighters stick to them



When the leaf-hopper punctures the plant the Blighter gets out for a ride



The leaf-hopper leaps to fresher feeding grounds, and carries a passenger

